Clubfoot in children

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Clubfoot, or talipes equinovarus, is a deformity consisting of equinus, varus, and adductus foot deformity. The true etiology of congenital clubfoot is unknown; several theories have been proposed. The pathology of the individual bones contributes to the clubfoot deformity and soft tissue contractures around the ankle and talocalcaneonavicular joint maintains the deformity and involve muscles, tendons, tendon sheaths, ligaments and joint capsules. Various treatment regimens have been proposed, including the use of corrective splinting, taping, and casting. Surgery in clubfoot is indicated for deformities that do not respond to conservative treatment by serial manipulation and casting. Surgery in the treatment of clubfoot must be tailored to the age of the child and to the deformity to be corrected.

The main goals of treatment is the painless, functional and anatomical normal foot without need for custom made footwear, and those can be achieved after detailed, individual approach with great experience in pediatric orthopedics.

Key words: clubfoot, nonoperative treatment, operative treatment

INTRODUCTION

Clubfoot, or talipes equinovarus, consisting of equinus, varus, and adductus foot deformity. This is one of the most common congenital orthopedic anomalies which continues to challenge the skills of the pediatric orthopedic surgeon because of tendency to relapse, no matter the foot was treated by conservative or operative means.

The incidence of clubfoot is approximately 1 case per 1000 and differs among ethnicities (it is close to 75 cases per 1000 live births in the Polynesian islands). The male-to-female ratio is 2:1; bilateral involvement is found in 30-50% of cases.

The equinovarus deformity is classified into congenital and acquired. The congenital is further classified into idiopathic and non-idiopathic types. The idiopathic type is an isolated skeletal anomaly, usually bilateral, has a higher response rate to conservative treatment and a tendency towards a late recurrence. The causes of the non-idiopathic type include deformity occurring in genetic syndromes, teratologic anomalies, neurological disorders of known (spina bifida) and unknown etiology and myopathies. The non-idiopathic type is characterized by diametrically opposite deformities in the feet (calcaneovalgus in one foot and equinovarus in the other), presence of other anomalies and a poor response to conservative or operative treatment. Acquired equinovarus has neurogenic causes (poliomyelitis, meningitis, sciatic nerve damage) and vascular causes. A postural deformity needs to be distinguished from a true clubfoot. The cause of the postural deformity is the position in utero in contrast to the true clubfoot, which has an underlying pathology, and the postural condition usually responds to passive manipulation by the parents.

ETYIOLOGY

The true etiology of congenital clubfoot is unknown. Several theories have been proposed regarding the cause of clubfoot. One is that a primary germ plasma defect in the talus causes continued plantar flexion and inversion of this bone, with subsequent soft-tissue changes in the joints and musculotendinous complexes. Another theory is that primary soft-tissue abnormalities within the neuromuscular units cause secondary bony changes. Other theories include: arrest of fetal development in the fibular stage, defective cartilaginous anlage of the talus, neurogenic factors, retracting fibrosis (or myofibrosis), anomalous tendon insertions, seasonal variations. Several authors have documented abnormal distribution of type I and type II muscle fibers in clubfeet.
Traditionally, two categories of club foot are identified: easy or correctable club foot (correct with manipulation, casting, and splintage alone) and resistant club foot that require surgery (responds poorly to splintage and relapse quickly following seemingly successful manipulative treatment).

The Pirani, Ginkel, or DeNegri classifications have been published, but no system is universally used. The Pirani scoring system can be used to identify the severity of the clubfoot and to monitor the correction.

PATHOANATOMY

The deformity varies in severity, however; the entire foot may be in an equinus and varus position with the forefoot adducted and a cavus deformity present, or the condition may be much less severe, with the foot being in only a mild equinus and varus position. Clubfoot is accompanied by internal tibial torsion. The ankle, midtarsal, and subtalar joints are all involved in the pathological process.

The equinus deformity is present at the ankle joint, talocalcaneonavicular (TCN) joint, and the forefoot. In the varus component, the hindfoot is rotated inward, primarily at the TCN joint. The whole of the tarsus, except talus, is rotated inward with respect to the lower leg. Since the forefoot follows the hind foot, the medial border of the forefoot faces upward. The adductus deformity takes place at the talonavicular and the subtal joints. The cavus component involves forefoot planter flexion, which contributes to the composite equinus. According to Turco, the talus is forced into equinus by the underlying calcaneus and navicular, whereas the head and neck of the talus are deviated medially. The calcaneus is inverted under the talus, with the posterior end displaced upward and laterally, and the anterior end displaced downward and medially. Mc Kay added the three-dimensional aspect of bony deformity of the subtalar complex in clubfoot so the relationship of the calcaneus to the talus is characterized by abnormal rotation in the sagittal, coronal, and horizontal planes.

The pathology of the individual bones contributes to the clubfoot deformity. The abnormalities of the talus include broadening of the anterior part of the tarsea, increased medial deviation and shortening of the neck, absence of the normal constriction of the neck and flattening of the talar head. The calcaneus is involved in all of the components of deformity and is grossly normal except that the three facets on the dorsal surface are flattened and the sustentaculum tali is hypoplastic. The navicular is displaced medially and its proximal concavity is flattened. The cuboid moves medially with the anterior end of the calcaneus and this causes the lateral convexity of the foot. The metatarsals also often are deformed and they may deviate at the phalangeal joints, or these joints may be normal, and the shafts of the metatarsals themselves may be adducted.

Soft tissue contractures around the ankle and talocalcaneonavicular joint maintain the deformity and involve muscles, tendons, tendon sheaths, ligaments and joint caps.

**FIGURE 1:**
TALOCALCANEAL ANGLE IN AP PROJECTION - NORMAL FOOT AND CLUBFOOT

The posterior contractures include the tendo Achille, tibialis posterior, tibialis anterior, peroneus longus, peroneus brevis, peroneus tertius, and posterior tibial. The calcaneocuboid ligament and calcaneocuboid liga ment. The lateral ligaments are involved in the talocalcaneal interossi ligament and the bifurcated Y ligament. The plantar contractures involve the adductor hallucis, plantar fascia and intrinsic toe flexors.

PRESENTATION

Clubfoot is often automatically assumed to be an equinovarus deformity but other combinations, such as calcaneovalgus, equinovarus and calcaneovalgus, are possible. In most of the cases, calcaneovalgus responds to conservative treatment, which involves passive manipulation by the mother and usually does not require casting or operative intervention.

Per anamnesis we should seek a detailed family history of clubfoot or neurovascular disorders. Clinical examination means general investigation to identify any other abnormalities and local foot status with the child prone (plantar aspect of the feet visualized), and aspire to evaluate internal rotation and varus. If the child can stand, determine if the foot is plantigrade, if the heel is bearing weight, and if it is in varus, valgus, or neutral. Idiopathic clubfoot is characterized by a deformed foot, prominence of the head of the talus, medial plantar eft, deep posterior eft, absence of normal creases over the insertion of Achilles tendon, calcaneal tuberosity situated at a higher level and atrophy of calf muscles. The three major components of the deformity, that is, equinovarus and plantar flexion, are obvious on examination. The attitude of the knee is usually flexed, but in cases of neglected clubfoot, the attitude of the knee will be hyperextension. The pres-
FIGURE NO 2:
TALOCALCANEAL ANGLE IN LATERAL PROJECTION - NORMAL FOOT AND CLUBFOOT

ece of other anomalies implies a non-idiopathic type of clubfoot, which has a poor prognosis.

Imaging studies generally are not required to understand the nature or the severity of the deformity \(^1\)\(^{,}\)\(^{31}\)\(^{,}\)\(^{32}\)\(^{,}\)\(^{33}\)\(^{,}\)\(^{34}\). Radiographs, however, are a useful baseline prior to and following surgical correction of the foot, closed Achilles tenotomy, or a limited posterior release. In a nonambulatory child, standard radiographs include anteroposterior and stress dorsiflexion lateral radiographs of both feet. Anteroposterior and lateral standing radiographs may be obtained for an older child. Important angles to consider in the evaluation of clubfoot are the talocalcaneal angle on the anteroposterior radiograph, the talocalcaneal angle on the lateral radiograph, and the talus-first metatarsal angle (Figures 1, 2). The anteroposterior talocalcaneal angle in normal children ranges from 30 to 55 degrees. In clubfoot, this angle progressively decreases with increasing heel varus. On the dorsiflexion lateral radiograph, the talocalcaneal angle in a normal foot varies from 25 to 50 degrees; in clubfoot, this angle progressively decreases with the severity of the deformity to an angle of 0 degrees. The tibiocalcaneal angle in a normal foot is 10 to 40 degrees on the stress lateral radiograph. In clubfoot, this angle generally is negative, indicating equinus of the calcaneus in relation to the tibia. Finally, the talus-first metatarsal angle is a radiographic measurement of forefoot adduction. In a normal foot, this angle is 5 to 15 degrees on the anteroposterior view; in clubfoot, it usually is negative, indicating adduction of the forefoot. Talocalcaneal paral-

leism is the main radiographic feature of clubfoot deformity.

With the advent of ultrasound, clubfoot can now be diagnosed at 18-20 weeks of gestation \(^3\)\(^{,}\)\(^{35}\); (only 80% accurate). Few studies of the ultrasonographic findings in normal feet or clubfoot have been performed, and the clinical utility of this modality has yet to be established. The main disadvantage of ultrasonography is the inability of the beam to penetrate all of the bones, particularly if a postoperative cast is present. Advantages of ultrasonography include the lack of ionizing radiation, no need for sedation, its ability to depict non-osseous portions of bones, and its capacity for dynamic imaging \(^3\)\(^{,}\)\(^{6}\)\(^{,}\)\(^{7}\).

TREATMENT

The initial treatment of clubfoot is nonoperative \(^3\)\(^{,}\)\(^{8}\)\(^{,}\)\(^{9}\)\(^{,}\)\(^{40}\). Various treatment regimens have been proposed, including the use of corrective splinting, taping, and casting. Treatment consists of weekly serial manipulation and casting during the first 6 weeks of life, followed by manipulation and casting every other week, until the foot is clinically and radiographically corrected. With experience, the orthopaedic surgeon is able to predict which feet would respond to nonsurgical treatment. The more rigid the initial deformity, the more likely that surgical treatment will be required. The order of correction by serial manipulation and casting should be as follows: first, correction of forefoot adduction; next, correction of heel varus; and finally, correction of hindfoot equinus. The Ponseti method consists of two phases: treatment and maintenance. The treatment phase should begin as early as possible, optimally within the first week of life. Gentle manipulation and casting are done weekly. Each cast holds the foot in the corrected position, allowing it to reshape gradually. Generally five to six casts are required to correct the alignment of the foot and ankle fully. At the time of the final cast, most infants require percutaneous Achilles tenotomy to gain adequate lengthening of the Achilles tendon.

Surgery in clubfoot is indicated for deformities that do not respond to conservative treatment by serial manipulation and casting \(^4\)\(^{1}\)\(^{,}\)\(^{42}\)\(^{,}\)\(^{43}\). Surgery in the treatment of clubfoot must be tailored to the age of the child and to the deformity to be corrected. In rare cases, for mild deformities with no severe internal rotational deformity of the calcaneus that requires extensive posterolateral release, the treatment of choice is a one-stage surgical release, such as the posteromedial release. A more extensive release that includes the posterolateral ligament complex most often is required for severe posterolateral deformity. The procedure described by McKay takes into consideration the three-dimensional deformity of the subtalar joint and allows correction of the internal rotational deformity of the calcaneus and release of the contractures of the posterolateral and posteromedial foot. Structures to be released or lengthened are the following: Achilles tendon, tendon sheaths of the muscles crossing the subtalar joint, posterior ankle capsule and deltoid ligament, inferior fibulobular ligament, talocalcaneal ligament, capsules of the ta-
CONCLUSION

Clubfoot deformity is seen in 1/1000 live births, bilateral in 50% of cases, with autosomal dominant inheritance and incomplete penetration. Etiology patterns differ from talus bone deficiency to neuromuscular abnormalities and secondary bone deformities. Pathoanatomy are located to ankle, subtalar and midtarsal joints with dominant deformities: equinus, valgus and adductus. There are different degrees and combination including cavus foot deformity as well as tibial torsion and muscle atrophies. Diagnostic criteria include clinical and radiographic examination which can help in treatment evaluation. Nonoperative treatment methods (manipulation, casts, orthosis or dynamic bands) as gold standard procedure, should start immediately after child birth to prevent structural changes. In resistant cases, or even in uncorrectable deformities in infants three months of age, surgery is the best choice of treatment. However, the main goals of treatment is the painless, functional and anatomical normal foot without need for custom made footwear, and those can be achieved after detailed, individual approach with great experience in pediatric orthopedics.

REZIME

KRIVO STOPALO KOD DECェ

Pes ekvinovanus (krivo stopalo) ili "clubfoot" je poremećaj koji se sastoji od ekvino, vanu i duktu deformitet stopala. Etiologija je nepoznata, a opisano je više različitih teorija. Patološke promene na pojedinim kostima doprinose deformitetu, a konstrukcije mekih tkiva, koje uključuju mišiće, tetive, tetivne omotnice, ligamente i kapsnula skočnog, talomavikularnog i subtalnog zgloba su dodatni faktori.

Oписани su različiti režimi neoperativnog lečenja, uključujući upotrebu korektivnih gipseva, ortoza i dinamičkih traka. Hinurgija je indikovana kod deformiteta koji se ne koriguju na konzervativno lečenje i mora se prilagoditi uzraza deteta i vrsti rezidualnog deformiteta.

Glavni cilj lečenja je bezbolesno, funkcionalno i anatomski normalno stopalo, bez potrebe za posebno prilagođenom obućom, što se može postići nakon detaljnog, individualnog pristupa i uz veliko iskustvo u pedijatrijskoj ortopediji.

Ključne reči: pes ekvinovanus, neoperativno lečenje, hirurško lečenje

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